U. S. DEPARTMENT OF AGRICULTURE
BUREAU OF ANIMAL INDUSTRY

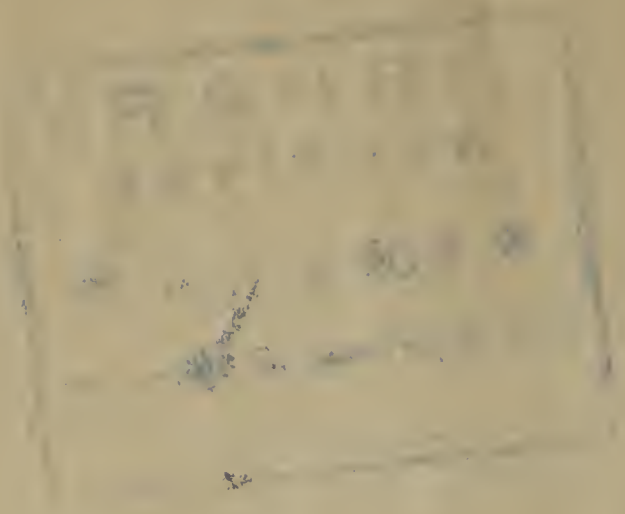
THE EFFECT OF SMELTER FUMES UPON THE LIVESTOCK INDUSTRY IN THE NORTHWEST

BY

ROBERT J. FORMAD, V. M. D., M. D.
Pathologist, Pathological Division

[Reprinted from the Twenty-fifth Annual Report of the Bureau of Animal Industry (1908), pp. 237-268.]

WASHINGTON : GOVERNMENT PRINTING OFFICE : 1926



THE EFFECT OF SMELTER FUMES UPON THE LIVESTOCK INDUSTRY IN THE NORTHWEST

By Robert J. Formad, V. M. D., M. D.,
Pathologist, Pathological Division

CONTENTS

INTRODUCTION

Have smelter fumes and emanations any injurious effect on livestock? Have such effects ever been recognized, demonstrated, and recorded? Such questions could be asked only by the uninitiated, or by those who reside outside the regions where smelters are operated. These disastrous conditions are only too well known to the inhabitants of smelter districts in any part of the globe, as well as to those familiar with the literature of the subject, who have substantial evidence, descriptive and ocular, upon which to base affirmative answers. Both animal life and plant life have succumbed to the smelters' baneful influences. Owners of livestock in regions where smelters are operated have suffered such losses among animals and forage that stock raising has had to be decreased or even abandoned; and no region adjacent to a smelter is exempt from the blighting effects of the fume-laden atmosphere upon vegetation, forests, and all forms of animal life. In fact these two industries, livestock and smelting, can not coexist in the same locality when the ores used in the smelters contain arsenic. This is said with full appreciation of the relative character of the damage perpetrated. The quantity and quality of the ore treated as well as topographical conditions will influence the damage inflicted; however, in every instance it is found that the effect is detrimental and that the loss sustained bears a ratio to the amount of ore handled.

In Europe the effects of smelter fumes upon the livestock industry have attracted the attention of investigators for more than a century, and the numerous data gathered by those workers have become history.

These earlier researches were directed principally toward the study of the damage to vegetation and forests from a botanical and chemical standpoint, the injuries to plant life being more readily seen than those to animal life. Forest culture and the preservation of forests have reached a high state of perfection and are zealously guarded by European governments, while the raising of livestock is on a smaller scale than ours, and the animals are mostly sheltered in stables.

In England, following the establishment of alkali works in 1796, such great losses were sustained that one company paid annually

to landowners and tenants for damages to trees, hedges, crops, etc. In 1820 the Swansea works were established and caused great damage, which was recognized and allowed by the courts in 1823 and again in 1832. In 1847 the towns improvement clause act was passed, in 1858 the local government act, later the smoke prevention act, then the smoke nuisance removal act, all of which bore upon this question.

In Germany, the valuable investigations by Stökhart in 1849, by Sussdorf in 1855, by Rosler in 1865, by Reich in 1867, by Freytag in 1870–1875, and by Haubner in 1878, are among the earlier scientific labors on this subject. The masterly descriptions by Freytag and Haubner of smelter-smoke conditions more than twenty-five years ago stand correct to-day almost without change. These works inspired the ablest scientists to investigate smelter conditions, and their results have corroborated Freytag's and Haubner's view that smelter fumes and flue dust are so injurious to the livestock industry that the two industries are incompatible in the same district. The more recent contributions on the injurious effects of smelter smoke are so numerous that they have been placed in the bibliography at the close of this paper.

The German Government has enacted laws whereby the smelting of ores is permitted only on smelter reserves; that is to say, the smelting company has to acquire all the land within the radius over which the injuries of the smelter fumes extend.

In the United States, whatever governmental control of smelting may exist, it is safe to say that hitherto in the laws enacted no account has been taken of the havoc wrought by the poison-laden fumes upon vegetation or animal life. Here forest culture and protection does not receive the same care and attention as in Europe, although smelting is conducted on a much larger scale and the ores contain more sulphur and arsenic than in Europe. The fact that our livestock is handled in larger flocks or herds makes it more difficult to handle individual animals, and thus a longer time has been required for the recognition of a diseased condition in the herd and its possible intimate connection with deleterious effects of the smelter. Hence it is that in this country only in scattered instances has this surmise culminated in an appeal to the courts.

OUTLINE OF PRESENT INVESTIGATION

The investigation described in this paper was undertaken by the direction of the Secretary of Agriculture in response to numerous appeals from stock growers and farmers of Deer Lodge Valley, Montana, who sustained such losses among their animals and crops that livestock raising not only became unprofitable, but a number of ranchers were compelled to abandon their homes and improved land and seek a livelihood elsewhere. The writer was detailed to inspect the conditions of that locality and to take such material from animal tissues for microscopic examination as might appear necessary.

The geographic situation of the Washoe smelter near Anaconda, Mont., and of the Deer Lodge Valley have been minutely described in Bulletin 113 of the Bureau of Chemistry of the United States Department of Agriculture; hence it is unnecessary to give such a description in the present article.

In this connection it is only proper to acknowledge the generosity of the Deer Lodge Farmers' Association in offering to allow the writer to select and slaughter without payment of compensation any animal—horse, cow, steer, or sheep—on any ranch in their so-called smoke zone, as well as from the dairies, to be utilized for the purpose mentioned. Mr. E. P. Mathewson, general manager of the Anaconda Copper Mining Co., likewise extended a similar offer with regard to any animals on the company's premises, consisting of section 16 and the leased part of the Bliss ranch. These offers were gratefully accepted.

The inspection of Deer Lodge Valley and the collection of material for microscopic examination during the winter of 1906 extended over a period of 44 days, from October 26 to December 8. It was ascertained first, by conversation with ranchers, what were the general conditions of the valley in which the losses were reported to occur, the extent of territory involved, the number of animals lost, the time of the year, and the localities where the greatest losses were sustained, also the character of the feed and water supply and the conditions under which the animals were kept. The representatives of the mining company, on the other hand, reported the flourishing condition of the animals on the company's premises, the abundant hay crops, etc.

The writer visited as many ranches as possible in the time mentioned, and inspected the pastures, soil, water supply, hay, sheds, and stables, as well as the physical condition of the available animals. This latter examination was usually made in conjunction with Dr. E. T. Davison, of the Bureau of Animal Industry; also on some occasions with Dr. D. E. Salmon, Dr. Leonard Pearson, and Doctor Cheney. The ranches inspected were located in various directions from the smelter, and their distance on an air line from the smelter varied from 1½ to 12 miles. While not every ranch came under observation, those that were investigated were thoroughly representative of the region subjected to the influence of flue dust. This region included almost the entire Deer Lodge Valley as well as the adjoining foothills. The injurious effects were sufficiently pronounced to be detected even on casual inspection, to say nothing of a close, careful, systematic investigation.

Twenty-one ranches were investigated, practically all of which were to the eastward of a line running north and south through the smelter; only one ranch was a little to the west of south, and situated about 4 miles from the smelter. Eight ranches were in a southeasterly direction, ranging from 1½ to 7 miles in distance; two were due east, from 2 to 7 miles; seven were northeast, from 3 to 9 miles; and the farthermost three were north, from 9 to 12 miles.

The inspection itself varied considerably in character. Where the owner was prejudiced against an investigation and unwilling to give information or have his premises and animals inspected the inspection was limited to a mere general inquiry as to the present state of hay and other crops harvested, the prices received for them, and the number of animals on hand as compared with the records of previous years. On the other hand, where the owner was disposed to sanction an investigation it took the form of a minute inspection of the ranch, pastures, hay, water supply, shelters, sheds, stable, and barn, in addition to an examination of all livestock. On some ranches the writer

was permitted to select and slaughter animals for the purpose of microscopic investigation, which must always be the court of last appeal in the consideration of damage to tissues.

It should be mentioned in this connection that the physical examination of range animals is not as easily accomplished as that of the eastern stable and pasture reared animals. When an animal is used to roaming on the range, even if it is sick, it does not submit as kindly to an examination as the halter-broken animal. After such preliminary examination it was possible to form an opinion independent of the hearsay statements of ranchers on the one hand and of the representative of the mining company on the other hand.

CONDITIONS FOUND IN REGION OF SMELTER

This inquiry and inspection revealed many interesting facts. In driving through the valley one could see apparently fine-looking pastures and meadows in which no stock had been allowed to run or from which no hay had been cut. The reason as explained by residents of the valley was that the grass was so noxious that it could not be used with safety or profit. Even the hay, they said, was unsafe to feed and also unprofitable for sale. It is a well-known fact that Deer Lodge Valley hay, when there is any demand at all, sells for about 50 to 75 per cent less than the hay from outside the smoke zone. In many places stacks of 100 to 250 tons remained for several years unsold and thousands of tons of hay remained uncut in various parts of the valley. The claims of the residents in regard to the noxious properties of the grass and hay can be substantiated by the chemical analyses of Dr. J. K. Haywood, of the Bureau of Chemistry, as shown in the following table taken from Bulletin 113 of that Bureau:

Arsenic content of forage in Deer Lodge Valley, expressed as arsenious oxid, as determined by J. K. Haywood, U. S. Bureau of Chemistry

[Calculated to dry basis]

Serial No.	Description of sample	Approximate distance and direction from smelter	Arsenious oxid per gram of dry sample	Arsenious oxid per 25 pounds avoirdupois of dry ration	Water-soluble arsenious oxid per gram of dry sample	Water-soluble arsenious oxid per 25 pounds avoirdupois of dry ration
			Milligram	*Grains*	*Milligram*	*Grains*
4114	Bunch grass	2 miles N	0.103	18.0	0.083	14.5
4106	Alfalfa	2½ miles N	.069	12.1	.041	7.2
4115	Pasture grass	3 miles N	.069	12.1	.028	4.9
4117	Range grass	do	.054	9.5	.034	6.0
4116	Pasture grass	4 miles N., Lost Creek.	.041	7.2	.020	3.5
4107	Red top	4 miles NE	.028	4.9	.014	2.5
4118	Clover	do	.054	9.5	.028	4.9
4119	Range grass	do	.090	15.8	.020	3.5
4120	Alfalfa and clover (just cut)	4½ miles NE	.054	9.5	.020	3.5
4108	Red top	5 miles NE	.055	9.6	.028	4.9
4121	Range grass	do	.090	15.8	.069	12.1
4122	do	6 miles NE	.104	18.2	.041	7.2
4123	do	8 miles NE	.055	9.6	.028	4.9
4109	Alfalfa	10 miles NE	.070	12.3	.042	7.4
4124	Red top	1 mile SE	.069	12.1	.041	7.2
4112	Bunch grass	3 miles E	.042	7.4	.020	3.5
4110	Field grass	6 miles E	.055	9.6	.041	7.2
4111	Hay	do	.041	7.2	.028	4.9
4125	Range grass	4 miles W	.055	9.6	.028	4.9
4113	Bunch grass	6½ miles W	.055	9.6	.028	4.9

The analyses of Prof. R. E. Swain, of Leland Stanford Junior University, California, as published in the Journal of the American Chemical Society, Volume XXX, No. 6, June, 1898, and Prof. W. D. Harkins, of the University of Montana, in 1906, given below, likewise show the presence of a large amount of arsenic in grass, hay, and leaves and bark of trees resulting from smelter fumes.

Arsenic content of grass, hay, etc., in Deer Lodge Valley, as determined by Prof. W. D. Harkins, University of Montana

Serial No.	Nature of sample	Month	Distance and direction from Anaconda	Parts per million [a]	Serial No.	Nature of sample	Month	Distance and direction from Anaconda	Parts per million [a]
			Miles					*Miles*	
38	Grass	February	5 SE	140	57	Grass	July	2 SW	508
39	do	do	5.5 N	180	58	do	do	0.7 SW	431
40	Hay	June	3 W	14	59	Hay	August	6 E	31
41	Grass	do	4 W	99	60	Bark	do	1.5 SW	300
42	Hay	do	3 E	107	61	Leaves	do	1.7 SW	683
43	do	do	4 E	18	62	Grass	do	1.7 SW	482
44	Grass	July	4.2 N	12	63	do	September	2.5 NW	81
45	do	do	8 NNE	111	64	do	do	2.5 SW	100
46	do	do	5 W	38	65	do	do	6 N	33
47	do	do	3 SE	21	66	do	do	4.2 N	34
48	do	do	1.5 SW	157	67	do	do	1 N	101
49	do	do	2 S	10	68	do	do	1 E	236
50	do	do	2 SW	359	69	do	October	10 SW	64
51	do	do	1.5 SW	460	70	do	do	13 SW	38
52	do	do	1.7 SW	293	71	do	do	35 N	29
53	Leaves	do	1.7 SW	583	72	do	do	34 N	21
54	Bark	do	1.5 SW	350	73	do	November	4.2 NNE	121
55	do	do	1.7 SW	376	74	do	do	6 NNE	73
56	Grass	do	6 N	18	75	do	do	1.5 E	705

[a] Parts per million are equivalent to ten-thousandths of 1 per cent, and multiplied by 0.7 give the number of grains in 100 pounds of substance.

The presence of such large quantities of arsenic in the grass and hay, as shown by the chemical analyses referred to, would fully account for Deer Lodge Valley hay being less readily sold than hay raised elsewhere. Such hay would be undesirable feed for animals, and from an economic standpoint it would appear wiser to let it remain uncut than to incur the expense of cutting and stacking, when, if disposed of at all, it would bring only about the cost of putting it up.

LOSSES OF LIVESTOCK

An attempt was made to ascertain approximately the extent of the losses in livestock sustained by the farmers in the smelter region during the period from 1902 to 1906. The writer endeavored to get as accurate a list as possible of the number of animals which each farmer had at the beginning and at the end of this period, and statements were obtained as to the holdings of 49 persons. The exact numbers were taken from books whenever possible, although most of the figures, especially for 1902, were necessarily estimates.

Considered in the aggregate, these reported losses were nothing short of appalling in magnitude. The notes show that out of a total of 2,447 horses in 1902 only 423 remained in 1906, and the losses in cattle were even greater. The decrease was said to be due almost entirely to deaths, as on account of the unthrifty condition of the animals there was practically no sale for them. Three men who undertook to raise sheep went out of the business, and five other

ranchers also left the valley on account of their losses during this period.

A striking instance is afforded by the claim of one of the largest stock raisers of Deer Lodge Valley, whose ranch was about 12 miles from the smelter. According to his itemized statement his total losses during 1903 amounted to $31,582. He lost by death during that year 34 horses, 101 cattle, and 800 sheep, and animals that did not die were greatly damaged. He also claimed to have sustained losses and damage due to slunk calves, loss of milk, and damage to hay, pastures, etc.

A HISTORIC FEEDING EXPERIMENT

The feeding experiment described below is inserted partly to show that the controversy regarding damage to livestock from smelter fumes is one of long standing, at any rate in Europe. The experiment took place in Germany forty-five years ago, and, as will be seen, was quite conclusive in its results. The work was carried out by a scientific commission under the personal supervision of Professor Stangel, the place being the agricultural academy farm at Tharand.[1] The object was to determine whether the forage in smelting regions produced any injurious effects on the health of cattle.

Two 6-year-old steers were used in the feeding trials, which lasted from June 7, 1864, to April 27, 1865. The experiment was divided into three periods. In the first period, from June 7 to August 20, the steers were fed in the following manner: One, the check animal, received daily 30 pounds of wholesome, sound hay, while the experimental steer received 30 pounds of hay that was pronounced to be damaged by smelter fumes to the extent of 40 per cent. During this period, lasting seventy-five days, no marked disturbances were noticed. The experimental animal consumed 29.68 pounds of hay daily, and increased in weight from 962 pounds to 1,025 pounds, or 63 pounds, which is an increase of 0.84 pound per day; while the check animal increased from 941 to 1,022 pounds, or 81 pounds, which is a daily increase of 1.08 pounds.

During the second period, lasting from August 21 to November 21, or ninety-three days, the experimental animal had diarrhea several times and the feces changed color and had an ill odor. The animal partook of but 25.62 pounds of hay daily and increased in weight from 1,003 to 1,072 pounds, which is a daily average of 0.74 pound. The check animal consumed the full ration of 30 pounds, and increased from 1,015 to 1,109 pounds, or an increase of 1.01 pounds per day.

Up to this point it appears that both animals gradually increased in weight, and that while the check animal continued to consume the maximum amount of hay per day, the experiment animal showed gradually less desire to eat.

The feeding experiments were next continued by Commissioner Engel for the third period of one hundred and fifty-seven days, from November 22 to April 27. During this time the experimental steer

[1] Reported by Freytag in "Jahrbuch für das Berg- und Hüttenwesen in Königreiche Sachsen auf das Jahr 1873." There is apparently a discrepancy in the weights of the experiment animals as given at the end of the first period and at the beginning of the second period the following day, which the present author is unable to explain. The figures have been correctly copied from the German report. Whatever error there may be is not sufficient to affect materially the value of the result.

consumed on an average but 21.4 pounds per day, and its body weight diminished from 1,070 to 1,045 pounds, which was a diminution of 0.17 pound per day. The check animal continued to eat 30 pounds of hay per day, increasing in weight from 1,109 to 1,210 pounds, which is an increase of 0.64 pound per day. On April 27 the check steer was sold, while the experiment animal was continued to be fed on hay from the smelter district from April 28 to November 8, or 195 days. The reluctance to eat this hay increased to such an extent that toward the end of the experiment the animal consumed daily but 14 pounds of hay, the average during the entire period being 16.6 pounds per day. The body weight diminished from 1,045 to 820 pounds, a daily loss of 1.15 pounds.

LESIONS PRODUCED EXPERIMENTALLY

The following experiment was made in order to test the effect of arsenic on horses and to compare the experimental results with the symptoms as found among animals in the vicinity of the smelter. The subject was a horse purchased at Rock Creek, Mont., in perfect health at the time of purchase and weighing about 850 pounds.

August 29, 1906, the animal was given 20 grains of white arsenic mixed with a little bran and water. August 30 it was given 40 grains of arsenic in solution mixed with the bran.

August 31 the animal appeared less bright, appetite not quite so good. Pulse, which had been full and strong, is not easily felt, owing to the bad disposition of the animal. The animal received again 40 grains of arsenic in solution as on the preceding day.

September 1. General condition unchanged; apparently normal amount of urine has been voided, and natural droppings. The animal again received 40 grains of arsenic in solution.

September 2. Animal is sick; no appetite; appears dull; walks very stiffly; droppings covered with thick, stringy, white mucus; respiration accelerated; pulse almost imperceptible; urine voided in small quantity; temperature 102.1° F.; staring coat. Forty grains of arsenic solution were mixed with bran and oats, but the animal would eat but a few mouthfuls and took not to exceed one-tenth.

September 3. Animal looks somewhat brighter, but has no appetite and walks in a stiff, uncertain manner; mucous membranes red; pulse almost imperceptible; temperature 100.8° F.; drinks very little. Forty grains of arsenical solution injected into the pharynx with a syringe.

September 4. Animal very dull and drowsy; has no appetite; drinks little; droppings very soft; visible mucous membranes much congested; submaxillary artery at the jaw is tender and full, but the pulse is weak, almost imperceptible, could not be counted; heart beats quick, jerky, but not hard, 87 per minute; respiration 21; temperature 100.7° F.; animal scarcely able to walk on account of progressive paralysis affecting the limbs, particularly the posterior ones. Forty grains of arsenical solution injected in the same manner as on the previous day, of which the animal swallowed about one-half.

September 5. Animal more drowsy and weaker than on the previous day; sways in walking; inclination to lift hind feet higher than usual rather than to drag them; mucous membranes very red; erosions above upper incisors; respiration easy, 20 per minute; heart beats quickly, 87 per minute; temperature, 99.8° F.; no signs of pain. The general appearance would indicate that the animal can live but a short time. No arsenic given that day.

September 6. Animal died at 10 a. m.

ARTIFICIAL PRODUCTION OF ARSENIC ULCERS

Dr. D. E. Salmon produced experimentally ulcers that were identical with the "sore nose" of horses in Deer Lodge Valley. The animal experimented upon was a horse kept in a stable at Para's ranch and not allowed to go out on the pasture. It was fed hay pur-

chased from outside the valley, so that there should be no question of any other influence. An arsenical paste was made by mixing white arsenic with water to a semiliquid consistency, and this was applied to the nasal septum with a little swab consisting of a tuft of cotton wrapped around the end of a stick. The details of the experiment follow.

October 3, 1906. Paste applied to nasal septum.
October 4. No visible effect; application repeated.
October 5. Irritation of the mucous membrane seen at the seat of application. No application.
October 6. Thickening of the mucous membrane; the appearance similar to the early stages of erosion preceding the formation of ulcers acquired by animals in pasture.
October 7. No application made.
October 8. The horse has a small sore where the arsenic was applied.
October 9. No application made.
October 10. The horse has a distinct sore in the nostril nearly half an inch in diameter. It is in the nature of a superficial erosion.
October 11. No application made.
October 12. The sore in the nostril has become larger; still superficial. Application continued.
October 13. Horse has two ulcers in the nostril which became confluent, also a fissure extending along the floor of the nostril and marking the course taken by the slight flow of the nasal secretion. The ulcers and fissure were similar to those seen in the nose of a horse which contracted ulcers naturally while in pasture, the animals having been brought together and the ulcers examined and compared.
October 14. Application repeated and the ulcer became more pronounced.
October 15. The ulcer is now one and one-half inches long by half an inch wide, covered with a thick black crust. Other crusts have formed on the skin and mucous membrane surrounding it. The sore is slightly moist, sufficient to make the dust adhere, but there is little, if any, suppuration. Where the moisture from the nostril has trickled over and dropped from the horse a fissure or superficial crack has formed below the ulcer.
This condition in every respect resembles the naturally formed ulcers in the noses of three other horses that have been in pasture at Para's ranch, where this animal was kept, even to the fissures below the ulcers.

Similar ulcers were produced by the application of flue-dust paste prepared and applied by Doctor Cheney to the nostrils of two horses that were also kept at Para's ranch.

CLINICAL SYMPTOMS

The clinical symptoms shown by the livestock in Deer Lodge Valley presented great variations, depending upon whether the animals subsisted exclusively on the valley forage or received additional feed raised outside of the smoke zone. Animals raised for the market were usually grazed all the year on the foothills. During the last few years it became necessary to bring them down to the valley and meadows during the winter months and feed hay. This in itself diminished the profit of stock raising. Taking into consideration the presence of a considerable amount of arsenic in the hay, as shown by chemical analysis, it becomes quite apparent that animals could not thrive on such feed; in fact, it would only be a question of time before they would become nonsalable and eventually die. Animals raised for utility, as horses and dairy cows, receive grain, bran, and hay raised outside of Deer Lodge Valley. Such animals show symptoms in a less pronounced manner than the range-reared animals.

The use of certain condition powders to counteract the effect of the valley hay appears to have had considerable success in experimental determinations, though, unfortunately, the facts were not made public and the community could not benefit by the results of those demonstrations. Lastly, some of the more valuable breeding animals (stallions) were fed exclusively on forage from outside the Deer Lodge Valley, which made their maintenance quite expensive, but such procedure was necessary to prevent illness and eventual loss, as was borne out by experience when this precaution was not adopted.

Another way in which the contaminated forage of the smoke zone has crippled, paralyzed, and in some parts completely wiped out the livestock industry in the Deer Lodge Valley is by bringing about failure of conception, abortion, and finally causing sterility. According to location, the losses from the failure of conception and abortion vary from 30 to 60 per cent.

SYMPTOMS IN CATTLE

The symptoms in cattle manifest themselves by general unthriftiness; languid, listless expression; eyes often inflamed; puffiness and tumefaction constantly present around the eyes, frequently accompanied by lachrymation and ophthalmia; dry muzzle; salivation; profuse mucous discharge from nostrils; pronounced garlicky odor of breath; ragged, shaggy coat; skin dry, brittle, and closely adherent to the adjacent parts ("hidebound"). The affected animal often droops its head; the back is frequently arched; tail retracted between hind quarters; abdomen tucked up; appetite impaired; there is loss of flesh and in many cases great emaciation; weakness of hind quarters; staggering; temperature elevated; pulse rapid, soft, and almost imperceptible; respiration shallow and hurried; fetid, profuse diarrhea; droppings dark in color, squashy or pulpy in consistency, and covered with mucus. Abortion is very common in pregnant animals, and frequent failure of conception ultimately results in sterility. Shrinkage in milk yield and finally complete suppression of lactation in the milk cow are not infrequent.

SYMPTOMS IN HORSES

The unthriftiness in horses (see fig. 29) is even more pronounced than in cattle, owing to the sanguinary temperament of the animals. The hair loses its natural luster, the animal appears shaggy, and the coat is often covered by tufts of hair that have not been shed for a year or more. The skin is dry, hard, and brittle, with extensive falling of hair, forming bald areas in the more chronic cases. The spirit and vigor of the animal are lost to a great extent, and the endurance is so impaired that even a moderate amount of exercise causes fatigue and profuse sweating. There are also weakness, loss of muscular coordination of hind quarters, and a stiff, stilty, staggering gait, frequently followed by paralysis of the hind limbs. The vision often becomes deficient, causing the animal to shy readily. Occasionally blindness is induced from ophthalmia of long standing and from ulceration and staphyloma of the cornea. Dilatation of the pupils and puffiness around the eyes are exceedingly common. Pronounced garlicky odor of breath is a constant manifestation. The gum line is very prominent, swollen, and of bluish-gray color.

The nasal ulcer so characteristic and prevalent among the horses running on pasture during the fall and winter months in the smoke zone of Deer Lodge Valley is seen in all stages of development. (See figs. 30 to 34.) It begins near the orifice, below the lachrymal duct on the side next to the septum, and appears to be produced by a severe local irritant. In the earliest stages the ulcer manifests itself as a small red area one-quarter to one-half inch in diameter. The mucous membrane is swollen at the seat of irritation; this is followed by an excoriation of the upper layers of the epithelium, then an erosion, and is finally succeeded by the formation of an ulcer, which may be so shallow as to involve only the epithelial lining of

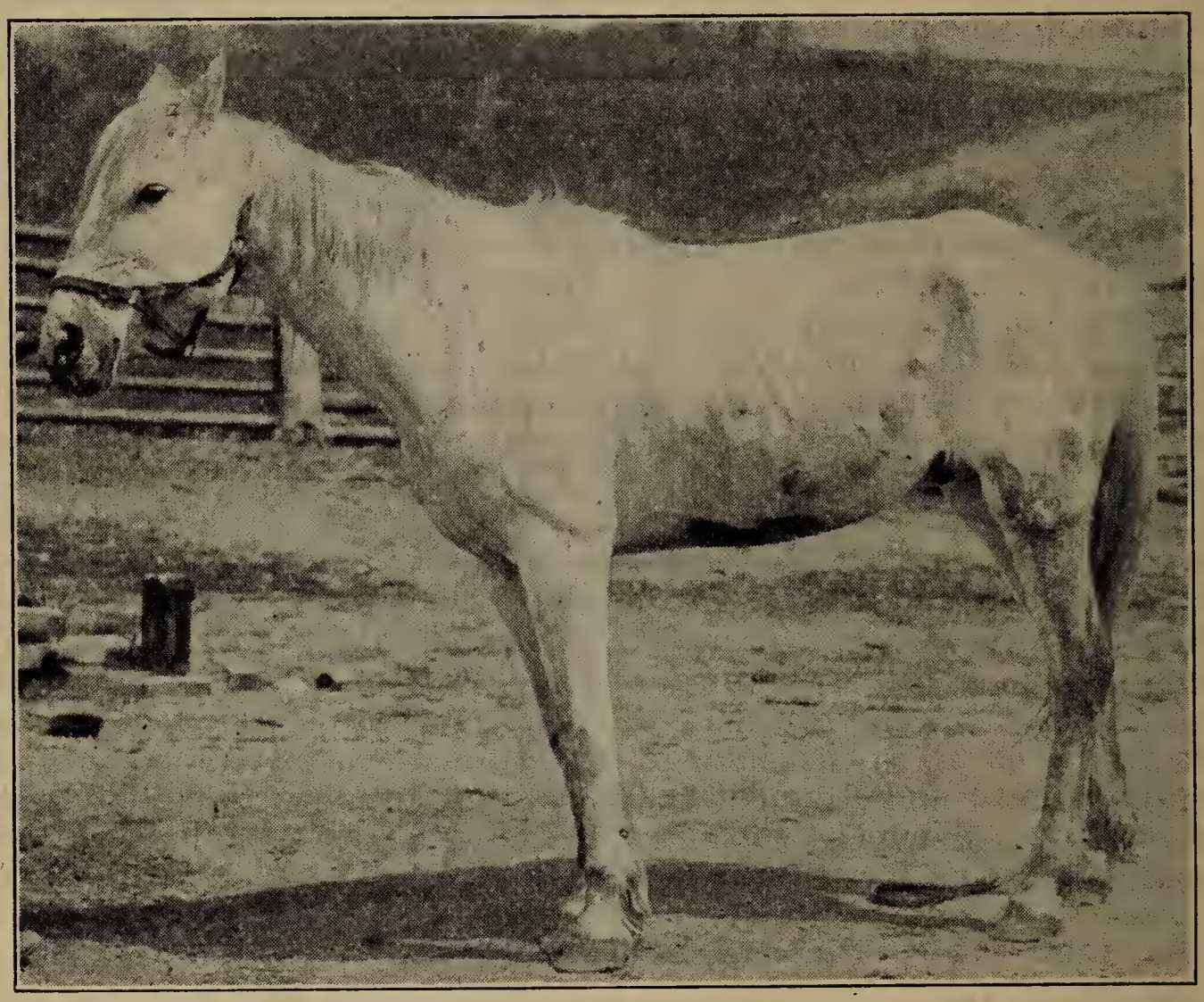


FIG. 29.—Horse showing general unthriftiness and several areas denuded of hair, due to arsenic poisoning from smelter. This animal was the last of 17 head, and died 10 days after picture was taken

the mucous membrane or may be deep enough almost to penetrate the nasal septum. The surface of the ulcer is covered by an inflammatory exudate, which is tightly adherent to the ulcer. The ulcer readily yields to treatment of the ordinary antiseptic solutions if the animal is stabled or corralled. Spontaneous recovery may also take place, as is indicated by the scars or cicatrices in the noses of many horses. The cicatrices frequently cause almost complete occlusion of the nostrils.

It has been suggested that the awns of foxtail produce these nasal ulcers, but such a supposition would be entirely out of the question, as the foxtail awns would more likely become lodged in the outer wall of the nasal passage than on the inner wall, where the characteristic ulcer is found. Besides, the suppuration around a foxtail awn is so infrequent, owing to the great vascularity of the part, and the

damage to the tissues so limited, that no true ulcer can be formed and the injured part heals without leaving a scar. On the other hand, the nasal ulcers in Deer Lodge Valley are identical with ulcers produced experimentally by the application of white arsenic and by flue dust by Doctor Salmon, and exactly resemble the slough produced from the application of white arsenic to warts, tumors, etc.

While the nasal ulcer is more prevalent during the fall and winter months, it may appear at any time. During the spring and summer, while the grass is growing rapidly and rains are frequent, not enough arsenic will accumulate on the vegetation to occasion very serious symptoms in animals grazing thereon or to cause the sore noses so prevalent at other seasons of the year.

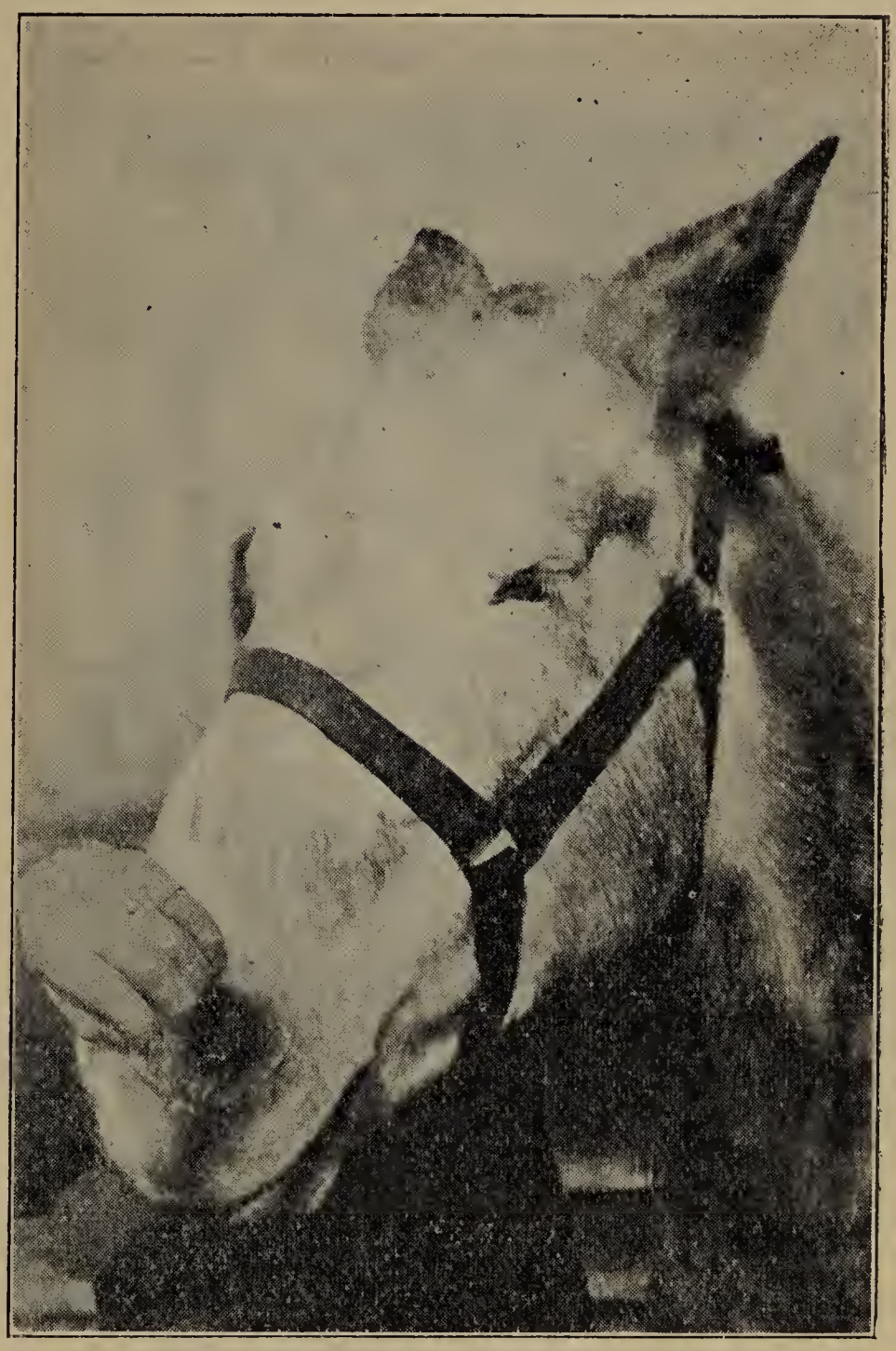


FIG. 30.—Filly with nasal ulcers as result of pasturing but a short time on a ranch about 10 miles northeast from smelter

POST-MORTEM NOTES

The object of the post-mortem examinations was to collect material for microscopic examination; hence only brief memoranda of the most salient lesions were made, rather than a detailed description of all the changes found. The total number of animals autopsied was 21, including 11 horses, 7 cattle, and 3 sheep. Seventeen of these animals were killed, while 4 had died. In addition, the remnants of a cow and fetal calf found by the roadside were examined.

Most of the tissues taken were fixed in Zenker's fluid. A limited number were fixed in Fleming's solution and some in a 10 per cent solution of formaldehyde. Four pieces were usually taken of the more important organs concerned in the elimination, as the kidneys, the liver, and the lungs, also of the stomach and the intestines, while only two pieces were taken of the organs less commonly affected, as the spleen, the heart, and the reproductive and nervous systems.

CASE 1

Colt, owned by Walter C. Staton. The animal was killed October 28, 1906, on Staton's lower ranch by shooting through the neck and bleeding. General condition unthrifty; much long hair; strong garlicky odor from nostrils; large ulcer in each nostril; gum zone prominent; trachea congested; lung congested, not resilient to touch; pleura thickened; heart firm; pericardium contained some yellow-colored effusion; abdominal cavity contained a considerable amount of transparent yellow effusion; stomach, characteristic appearance found in affected animals in Deer Lodge Valley, namely, reddish brown in color, walls thickened and in a state of chronic catarrh; serous surface of small intestine pale, mucous membrane congested; liver, greenish bile discoloration; spleen enlarged and pigmented; kidneys enlarged and congested; brain apparently normal; fat more yellow than normal.

FIG. 31.—Horse with nasal ulcers which developed while pasturing on a ranch about 4 miles northeast from smelter

The following tissues were taken for microscopic examination: Ulcer from nose; lung, liver, kidney, heart, spleen, these being placed in Zenker's fluid; cerebrum and cerebellum put in 10 per cent formaldehyde.

CASE 2

Bay gelding, aged 3, owned by Thomas Elliott. Animal killed November 2, 1906, by shooting through the neck and bleeding. General condition unthrifty; strong garlicky odor from nostrils; prominent gum line; ulcer in left nasal orifice; trachea congested; lungs slightly catarrhal and emphysema in anterior tips of both lobes; heart hypertrophied; stomach in a state of chronic catarrh, a few bots present at the pyloric orifice; small intestine slightly catarrhal; duodenum contained a countless number of bots; large intestines contained a few sclerostomes; liver congested and bile-stained; both kidneys congested and enlarged; suprarenals enlarged; spleen enlarged; pancreas hypertrophied; bladder congested.

Tissues taken for microscopic examination: Lung, liver, kidneys, stomach and pancreas, heart, bladder and adrenal, spleen, these being placed in Zenker's fluid; kidney and liver in Fleming's solution.

CASE 3

Bay gelding, owned by Nels Pierson. Animal killed November 2 on Staton's lower ranch by shooting through the neck and bleeding. General condition unthrifty; edema around eyes; marked garlicky odor from nostrils; prominent gum line; ulcer in nasal orifice; trachea congested; lungs apparently normal, except a few small congested areas; heart hypertrophied, very firm; stomach

in a state of chronic catarrh; liver apparently normal on microscopic inspection; spleen enlarged and greatly pigmented; both kidneys slightly congested, about normal in size; pancreas, adrenals, and bladder apparently normal.

Tissues taken for microscopic examination: Lung, liver, kidneys, heart, stomach, spleen; all placed in Zenker's fluid.

Case 4

Bay gelding, owned by N. Bielenberg. Animal killed November 4, 1906, on P. Lappin's ranch, by shooting through the neck and bleeding. General condition unthrifty; strong garlicky odor from nostrils; puffy swelling around eyes; prominent gum line; erosion in both nostrils, but no ulceration; trachea congested; lungs have mottled appearance from patches of congestion with the tips of anterior lobes emphysematous and atrophic; pleura greatly thickened; heart hypertrophied and fairly soft in consistency; endocardium thickened; stomach inflamed, coated with mucus, and in state of chronic catarrh; small intestines congested in places, especially the jejunum, which had a number of congested areas 1 to 1½ inches in size; liver hypertrophied, bile-stained, and covered by a goodly number of tufts; the fat throughout the body was very yellow in color; spleen enlarged and pigmented; kidneys congested, catarrhal; adrenals enlarged; pancreas and salivary glands, especially the parotid, were edematous; bladder congested; brain and cord congested; a considerable amount of cerebrospinal fluid was present.

FIG. 32.—Colt with extensive nasal ulcers of recent origin. This colt was unbroken and was cast in order to get photograph

Tissues taken for microscopic examination: Lung, pancreas, adrenals, and liver, kidney, heart, stomach, intestines, in Zenker's fluid; cerebrum and cerebellum, spinal cord, and eye in 10 per cent formaldehyde.

Case 5

Remnants of cow and fetal calf by the roadside, about a quarter of a mile from P. Lappin's ranch, were examined November 9, 1906. The organs appeared somewhat abnormal, especially the stomach and liver, which were congested; spleen enlarged; kidneys not present.

Tissues taken for microscopic examination: Liver, stomach, spleen, ovary, in Zenker's fluid; stomach and fetal tissues in 10 per cent formaldehyde.

Case 6

Calf about 6 months old, owned by P. Lappin. Animal killed November 9, 1906, by bleeding, on Lappin's ranch. General condition unthrifty, emaciated; trachea slightly congested; lungs apparently normal, anemic; all of the abdominal viscera anemic; the first three stomachs contained a fair amount of food and were apparently normal, as was the fourth stomach; small and large

intestines pale but otherwise normal; mesenteric lymph glands considerably enlarged; liver, kidneys, spleen, pancreas, and reproductive organs apparently normal.

Tissues taken for microscopic examination: Lung, liver, kidney and lymph glands, stomach, ovary, in Zenker's fluid; lymph glands in 10 per cent formaldehyde.

Case 7

Sheep owned by Deer Lodge Farmers' Association. Animal killed November 11, 1906, by bleeding, on Para's ranch. General condition fair; trachea congested; lungs anemic, apparently normal; fourth stomach considerably congested; small intestines contained a number of inextensive, slightly congested areas; heart, liver, spleen, and glands apparently normal; kidneys slightly congested and enlarged.

Fig. 33.—Horse showing pronounced nasal ulcers. The animal was pastured but a short time on a farm distant about 3½ miles northeast from smelter

Tissues taken for microscopic examination: Lung, kidney, stomach, heart, spleen, all in Zenker's fluid.

Case 8

Idaho steer, owned by the Anaconda Copper Mining Company. Animal killed November 16, 1906, on section 16, company's ranch, by shooting and bleeding. General condition of animal very good; mucous membrane of trachea congested; lungs apparently normal; first three stomachs apparently normal, while fourth stomach was slightly congested; small intestines apparently normal, with the exception of enlargement of the Peyer's patches and the mesentric glands. A few small areas of localized congestion were present in the jejunum. Liver, heart, spleen, pancreas, and salivary glands apparently normal; adrenals enlarged; kidneys slightly congested; bladder apparently normal. An ulcer on the anterior portion of the tongue was probably of traumatic origin.

Tissues taken for microscopic examination: Lung, liver and pancreas, kidney, stomach, intestines, heart, spleen, mucous membrane from trachea, ulcer from tongue, in Zenker's fluid; intestines in 10 per cent formaldehyde.

Case 9

Black cow, owned by George Cammack. Died and autopsied on Cammack's ranch while the carcass was still warm, November 17, 1906. General condition quite good; trachea congested; lungs congested, inflamed, and adherent to walls of chest in several places; pleura greatly thickened; pericardium greatly thickened and excessively distended by dark-brownish liquid of foul odor; heart hyper-

trophied; abdomen contained a large quantity of transparent yellowish liquid; liver very large, weighing 25 pounds, very firm in consistency, stained by bile pigments, and in a state of hypertrophic cirrhosis and streaked with blood; fourth stomach intensely inflamed, several small necrotic areas being present; both small and large intestines intensely inflamed throughout, walls greatly thickened from accumulation of transparent liquid between serous and muscular coats; kidneys presented a mottled appearance of deep red and light areas on the surface, while on section several anemic infarcts were present in the medulla of the organ; adrenals enlarged; pancreas hypertrophied; bladder inflamed.

Tissues taken for microscopic examination: Lung, liver, kidney, stomach, heart, pericardium, all in Zenker's fluid.

FIG. 34.—Mare showing healing ulcers in nostrils. Animal was taken from pasture and treated locally with carbolic acid and sweet oil. Distance about 9 miles north from smelter

CASE 10

Brown-and-white cow, owned by John Furst. Animal killed November 18, 1906, by shooting and bleeding on Furst's ranch. General condition fair; trachea congested; lungs crepitant and apparently normal; heart flabby, some effusion in pericardium; true stomach slightly congested; both small and large intestines showed limited congested areas; liver had marked cirrhosis, was bile-stained, and had a number of tufts on the surface; kidneys slightly congested in cortical portion; spleen enlarged; pancreas, adrenals, and bladder apparently unaffected.

Tissues taken for microscopic examination: Lung, liver, kidneys, stomach, heart, spleen, all in Zenker's fluid.

CASE 11

Bay mare, owned by John Furst. Animal killed November 18, 1906, on Furst's ranch by shooting through neck and bleeding. General condition fair. This animal had previously broken its left fetlock and was hobbling on three legs. Trachea congested; lungs congested; heart slightly enlarged; marked congestion of the stomach. Both small and large intestines were congested, more particularly the former, which were in a catarrhal condition. Pancreas apparently unaffected; kidneys congested; adrenals enlarged; spleen congested and enlarged; bladder slightly congested.

Tissues taken for microscopic examination: Liver, kidney, stomach, all in Zenker's fluid.

CASE 12

Horse, owned by State Senator Harper. Spontaneous death; animal autopsied on city dump November 26, 1906. General condition fair; trachea congested; pronounced congestion and inflammation of lungs; pleura thickened; stomach inflamed, in state of chronic catarrh; both small and large intestines intensely congested; liver hypertrophied, congested, with abundant connective tissue visible to the naked eye; spleen enlarged and pigmented.

Tissues taken for microscopic examination: Lung, kidney, stomach, intestines, stomach, all in Zenker's fluid.

CASE 13

Sheep, owned by N. Bielenberg. Spontaneous death; autopsied on Bielenberg's ranch November 27, 1906. General condition fair; no evidence of decomposition; trachea congested; lungs and heart apparently normal; fourth stomach decidedly congested and inflamed; both small and large intestines congested; there were also some localized areas of inflammation in the small intestines; liver, spleen, and pancreas showed no microscopic lesions.

FIG. 35.—Sheep with ulcers in the nose. Out of a flock of 60 pastured about 10 miles north of smelter, 10 were examined, and 3 of these had nasal ulcers

Tissues taken for microscopic examination: Liver, kidney, stomach, intestines, heart, and lung, all in Zenker's fluid.

CASE 14

Aged roan cow, owned by Anaconda Copper Mining Co. Animal killed December 1, 1906, by shooting on company's ranch. This cow had been in Anaconda Copper Mining Co.'s possession since October, 1905; purchased out of Staton's stock; had been on pasture about 4 miles northeast of smelter. General condition fair, but not as good as the Idaho animals; pronounced garlicky odor from nostrils; mucous membrane of trachea red and congested; lungs apparently unaffected; heart, some thickening of endocardium, myocardium apparently unaffected; first three stomachs apparently unaffected, fourth stomach somewhat congested, but not excessively; liver enlarged, showed hypertrophic cirrhosis; spleen pigmented, with several localized areas of induration; small intestines slightly congested; kidneys congested at the cortex; pancreas, adrenals, and bladder showed no microscopic lesions.

Tissues taken for microscopic examination: Lungs, kidneys, stomach, intestines, spleen, liver, in Zenker's fluid; liver and kidney in Fleming's solution. Samples of liver and kidney were also taken for chemical examination.

CASE 15

Red heifer, owned by P. Lappin. Animal killed December 3, 1906, on Lappin's ranch by knocking and bleeding. General condition unthrifty, anemic; trachea slightly congested; lung pale, shrunken, but no apparent macroscopic lesion; heart flabby; stomach apparently unaffected; small intestine slightly congested; liver hypertrophied, pale, rather soft in consistency; kidneys decidedly mottled, most pronounced areas of degeneration, atrophy of cortex; adrenals, spleen, pancreas, and bladder showed no macroscopic lesions.

Tissues taken for microscopic examination: Lung, liver, kidney, stomach, heart, spleen, in Zenker's fluid; liver and kidney in Fleming's solution.

Case 16

Aged dark-brown horse, owned by L. Leffering. Animal killed on P. Lappin's ranch December 3, 1906, by shooting through the neck and bleeding. General condition unthrifty; pronounced garlicky odor from nostrils; ulcer in right nostril; lungs congested, emphysematous around lower border and atrophic at tips and lobes; heart hypertrophied; considerable amount of effusion in abdominal cavity; mucous membrane of stomach swollen and inflamed at the pyloric portion; small intestines decidedly congested, particularly in the upper portion; few bots in duodenum; liver congested, enlarged, firm; cortex of kidney very congested, glomeruli prominent; spleen congested, deeply pigmented; adrenals enlarged; bladder apparently unchanged.

Tissues taken for microscopic examination: Lung, liver, kidney, stomach, heart, spleen, in Zenker's fluid; liver and kidney in Fleming's solution; liver and kidney and nasal ulcer in 10 per cent formaldehyde.

Case 17

Brown filly, owned by Anaconda Copper Mining Co. Animal killed December 4, 1906, on section 16, company's ranch, by shooting and bleeding. General condition fair; pronounced garlicky odor from nostrils; no nasal ulcer; marked gum line; trachea slightly congested; lungs apparently unaffected; pleura thickened; heart normal; mucous membrane of stomach thickened, inflamed, and in a state of chronic catarrh. External examination of intestines revealed the presence of a large aneurism of the anterior mesenteric artery, caused by sclerostomes. On opening the small intestines, the mucous coat was found slightly congested. The cecum contained a large number of sclerostomes and was filled with digested food and a large amount of very fine brownish-black seeds which the animal could not have gotten on the pasture, but were probably administered for medicinal purposes. Liver was slightly sclerotic and enlarged. A moderate amount of congestion was found in the cortical portion of the kidney. Adrenals, pancreas, spleen, and bladder apparently unaffected on macroscopic examination.

Tissues taken for microscopic examination: Lung, liver, kidney, stomach, heart, spleen, aneurism, in Zenker's fluid; aneurism in 10 per cent formaldehyde solution.

Case 18

Iron-gray horse about 3 years old, owned by Anaconda Copper Mining Co. Animal killed December 4, 1906, on section 16, company's ranch, by shooting and bleeding. General condition good, except some interference with breathing after exercise. Pronounced garlicky odor from nostrils; marked gum line; trachea slightly congested; lungs apparently unaffected; mucous membrane of stomach thickened, in a chronic catarrhal state, covered with mucus; mucous membrane of the small intestines slightly congested; cecum contained a limited number of sclerostomes and a large amount of fine brownish-black seeds intermingled with digested and macerated food. There was also an aneurism of the mesenteric artery, but not as large as in the preceding case. Liver stained by bile pigment; kidneys slightly congested; adrenals enlarged; pancreas, spleen, and bladder showed no macroscopic lesions.

Tissues taken for microscopic examination: Lung, liver, kidney, stomach, duodenum, heart, all in Zenker's fluid.

Case 19

Sheep, owned by Deer Lodge Farmers' Association. Animal killed by bleeding on B. Para's ranch, December 5, 1906. General condition good. The animal had been previously examined in November. Marked gum line; mucous membrane of trachea decidedly congested; lung slightly congested; heart flabby; first three stomachs normal; mucous membrane of fourth stomach congested at pyloric orifice; both small and large intestines showed no macroscopic lesions; liver slightly congested; remaining organs showed no macroscopic lesions.

Tissues taken for microscopic examination: Liver, kidney, lung, intestines, stomach, all in Zenker's fluid.

Case 20

Gray gelding, owner unknown. Animal broke its leg and was killed by shooting and bleeding on B. Para's ranch, December 5, 1906. General condition unthrifty;

trachea congested; lungs congested; stomach inflamed, chronic catarrh; small intestines slightly congested; large intestines contained few sclerostomes; small aneurism of the small mesenteric artery; liver slightly congested; kidneys congested; spleen pigmented and enlarged; pancreas, adrenals, and bladder showed no apparent macroscopic lesions.

Tissues taken for microscopic examination: Lung, liver, kidney, intestines, in Zenker's fluid; aneurism in 10 per cent formaldehyde.

Case 21

Young bull, owned by Bert Para. Animal killed on Bliss ranch, December 6, 1906, by shooting and bleeding. General condition unthrifty; trachea slightly congested; lungs and heart apparently normal; mucous membrane of stomach slightly congested; both small and large intestines showed no apparent macroscopic lesions; slight congestion of cortex of kidney; adrenals, spleen, pancreas, and bladder apparently normal.

Tissues taken for microscopic examination: Lung, liver, kidney, stomach, spleen, testicle, in Zenker's fluid.

Case 22

Bay mare, owned by Mathews Smith. Autopsied December 7, 1906, about thirty-six hours after spontaneous death. General condition, large animal in good flesh, bruised extensively. Abdomen greatly distended by gases; lung and heart greatly congested; stomach and intestines intensely congested, marked putrefactive changes. The small quantity of intestinal contents present was liquid in consistency. There were also marked traces of scouring on the hind quarters. Kidneys intensely congested, soft, and decomposed; liver congested, bile-stained, and soft; bladder congested; mucous membrane of uterus and vagina congested and edematous.

No tissues were taken for microscopic examination, but samples of stomach, intestines, kidney, and liver were taken for chemical examination.

The organs from two deer, shot by Mr. Cheney Beal on the foothills of Deer Lodge Valley, northeast of the Elliott ranch, were also examined and samples taken for chemical examination. The stomachs, livers, and kidneys were intensely congested and inflamed.

From the 21 autopsies 135 samples were taken. Out of this number 20 samples were selected from the more typical cases for microscopic study and description.

MICROSCOPICAL ANATOMY

Case 1

Slide A.—From mucous membrane of colt's stomach. The thickened and swollen mucous membrane shows no erosion or necrosis. The interglandular capillaries are greatly distended by blood at various points, particularly in the superficial portion of their course. In some places several capillaries are grouped so closely together that they correspond to petechiæ or hemorrhagic points, which were seen distinctly on macroscopic inspection at the autopsy. The chief and parietal cells show no degenerative changes. At the bottom of the mucosa in the substantia propria or stroma a slight inflammatory round-cell infiltration is present and extends at various points between the gastric glands, but is more abundant around the blood vessels. Such cell infiltrations have been originally described by Virchow as gastroadenitis arsenicalis parenchymatosa. The muscularis mucosa is slightly thickened, the entire mucosa is greatly hypertrophied and congested, and the walls of the hyperemic blood vessels are thickened. The surface of the organ is coated by a thin layer of mucus, fissured or broken in several places, and is infiltrated by hemorrhages.

Slide B.—Section from cortex of kidney. The walls of the capillaries are thickened and distended by blood. There is also an increase in the supporting connective tissue as well as a proliferation of the connective-tissue cells. The glomeruli are slightly congested. Diapedesis of red blood corpuscles is seen between the proximal and distal convoluted tubules of the labyrinth. Several small hemorrhagic areas are present in the middle of the cortex. The cytoplasm of the renal epithelium lining the convoluted tubules is quite granular, which condition in some parts is sufficient to suggest cloudy swelling. Occasional necrotic areas and vacuolation are present where the change has advanced to a state of

fatty degeneration. The karyoplasm is also in varying degrees of degeneration, so slight in some cells as to appear almost normal and taking the staining quite well, while in other cells it has become quite irregular and is in a state of karyorhexis or fragmentation or in a state of karyolysis or dissolution, when it has disappeared either partly, where the nuclei stain poorly, or altogether, when it is impossible to distinguish the nuclei. The vacuolation of the cytoplasm is the turning point from advanced cloudy swelling to fatty degeneration, followed in places by minute necrotic areas which subsequently liquefy, the result of liquefaction necrosis bringing about vacuolation of the cytoplasm.

Slide C.—This is also a section from the kidney. The piece was taken from the side instead of the upper part of the organ and comprises more medullary portions than cortical. The collecting tubules being cut almost at right angle to their course, the congestion in this part of the kidney is even more extensive than in the cortical region. The distended capillaries each form areas smaller in size, but they stand out in greater contrast than in the cortex on account of structural peculiarities of the collecting tubules.

Slide D.—Section of kidney taken from the medulla and cut in the axis of the collecting tubules as far as possible, where near the tip of the renal papillæ the ducts of Beleni are cut transversely. The most notable feature of this preparation is the partial loosening of the epithelium in masses from the support and the absence of degenerative changes, which can be attributed to the straight course of the collecting tubules. The numerous capillaries, however, are somewhat distended, which condition becomes more pronounced as we ascend from the renal papillæ toward the boundary layer, where some of the capillaries assume more the nature of a hemorrhagic area from the close proximity of the vessels than that of mere distended vessels. The connective-tissue cell proliferation in the stroma of the medulla is not as pronounced as between the convoluted tubules of the cortex and in the vicinity of the renal capsule; still there are enough inflammatory cells present to indicate a decided inflammation of the organ.

Slide E.—Section of lung. The pleura is thickened and all the pulmonary capillaries are greatly congested and in some places distended to the point of engorgement, which becomes the characteristic feature of the preparation. There is a moderate amount of inflammatory cell proliferation in the peribronchial connective tissue, though less pronounced in the interalveolar tissue. Small circumscribed areas of the alveoli are filled with a moderate amount of catarrhal exudate containing a few desquamated pulmonary epithelial cells, but the process is not extensive enough to be of much consequence and is mentioned rather as descriptive evidence of its presence.

Slide F.—Section of liver. The characteristic feature in this section is the hypertrophic cirrhosis with proliferation of bile ducts. The liver lobules, the outlines of which are traced with difficulty in the normal organ, are here well separated from one another by large masses of proliferating young growing connective-tissue cells. This cell proliferation is most conspicuous in the periportal areas, which appear to be the primary and principal seat of the operation of the irritant, brought there from the intestines by the portal circulation. The proliferation of the bile ducts appears also to be the result of action of the same irritant. The cytoplasm of the hepatic cells is not profoundly altered, although some of the cells are quite granular, stain faintly, and stand out in great contrast to the less affected cells that have taken the staining quite well. In many of the liver cells a small amount of pigment is present. A moderate amount of outwandered leucocytes and diapedesis of the red blood corpuscles is present in the vicinity of the capsule of Glisson, extending also for some distance into the interior of the organ, where the corpuscles are aggregated sufficiently dense to form blood areas. There is also a good-sized hemorrhage near the capsule of Glisson. This hemorrhage should not be confounded with a neighboring portal vein which is more extensive than the hemorrhage.

Slide G.—Section of cardiac muscle. The endocardium is considerably thickened. The individual fibers in the myocardium are not materially altered by degeneration, though there is a greater number of connective-tissue cells than is generally found between the cardiac fibers. There is also a moderate amount of leucocytes and diapedesis of red blood corpuscles seen to a better advantage in places where the fibers are cut longitudinally.

Case 2

Slide A.—Section of stomach. The mucous membrane shows erosions in three different places where the surface epithelium has been denuded. The cells lining the ducts of the glands are desquamated in many places. The des-

quamation is not limited to the ducts alone, but involves about one-third of the gland immediately adjacent. The gastric epithelium shows no marked degeneration except an increased granulation of the cytoplasm of the central cells. Many interglandular capillaries are more congested than in the preceding case, while the inflammatory cell proliferation at the bottom of the mucosa is also present in this case, but to a less degree than in the previous one. The submucosa is greatly hypertrophied and all the capillaries are congested. The muscularis and the serous coat are also hypertrophied, but show no other alteration.

Slide B.—Section of kidney comprising both cortex and medulla, stained in picro-fuchsin. The capsule is thickened and is bloodstained on the outer surface, where the blood corpuscles tightly adhere and apparently penetrate the outer surface of the capsule. On the inner surface of the capsule the extravasated blood is accumulated in several places and permeated between the uriniferous tubes quite extensively for some distance into the interior of the cortex. The capillaries throughout the cortex are congested, particularly in the vicinity of the Malpighian bodies. All glomeruli are likewise congested, except those situated in the columns of Bertini. Outwandered leucocytes and diapedesis of the red blood corpuscles and hemorrhagic areas of limited size are present in various places. The connective-tissue cell proliferation present in the stroma is very abundant in the region of the boundary layer and only slightly in evidence in other parts of the kidney. The epithelium lining the proximal and distal convoluted tubules in the labyrinth is granular, degenerated, and desquamated, whereas in the collecting tubules these changes are not so apparent. The medulla of the kidney is also congested, and the congestion is even more disseminated than in the cortex.

Slide D.—Section of liver. The capsule of Glisson is thickened. There is a slight capillary hemorrhage in some of the lobules, also an increase of the interlobular connective tissue, which has contracted and partly distorted the liver lobules, and constitutes the atrophic cirrhosis which is the characteristic lesion in this preparation. The cirrhotic process in this liver differs very materially from that in case 1, where it is hypertrophic in nature, with cell proliferation, whereas in this case, instead of the cells, there are present connective-tissue fibers that have contracted, constituting the atrophic form. A limited amount of fat situated at the periphery of some of the lobules is in the nature of an infiltration rather than a degeneration. The connective-tissue cell proliferation around the portal canals, while present, is very slight. The hepatic cells are quite granular, some are atrophic, while others show an advanced state of cloudy swelling but no vacuolation. The cell nuclei have suffered either fragmentation or dissolution, the karyoplasm staining so faintly that it can not be distinguished from the cytoplasm of the cell. There is also a slight bile pigmentation present in many of the hepatic cells.

Case 3

Slide A.—Section of stomach. The mucous membrane of the stomach shows two distinct areas of erosions where the surface cells have undergone necrosis, thus forming small cup-shaped depressions, the edges of which are regenerating. The interglandular capillaries are congested, moderate outwandering of leucocytes and diapedesis of the red blood corpuscles are present, and numerous hemorrhagic areas may be seen at various levels of the greatly hypertrophied mucosa. The epithelium lining the ducts, as well as that lining the gastric glands for about one-quarter of its extent adjacent to the ducts, is somewhat degenerated and often desquamated. There are pronounced outwandering of leucocytes and diapedesis of the red blood corpuscles in the submucosa, which is likewise hypertrophied. The remaining coats of the stomach show no decided alteration.

Slide B.—Section of kidney from the medulla cut obliquely to the axis of the uriniferous tubules. The preparation shows an extensive congestion of all the capillaries. Neither degeneration nor desquamation of the renal epithelium is present in this section. (Subsequent examinations of other sections from the cortex of this kidney showed both degeneration and desquamation present in that portion of the organ, but not as pronounced as in case 1.)

Slide D.—Section of liver. The characteristic lesion in this section is the hypertrophic cirrhosis, which is even more pronounced than in case 1. The connective-tissue cell proliferation is so extensive as to outline perfectly nearly every liver lobule. The periportal inflammatory cell proliferation is less extensive than in case 1. The cytoplasm of the majority of the hepatic cells is swollen and granular, this latter condition constituting cloudy swelling, vary-

ing in intensity. The karyoplasm has also suffered decided alteration of the chromatin either by fragmentation or by dissolution, which has diminished the staining property of the nucleus. Neither necrosis nor vacuolation has taken place in the cytoplasm of the cells.

Case 8

Slide A.—Section of steer's stomach near the pyloric portion. The mucous membrane shows no erosion, and the cells lining the surface have not been detached. The epithelium lining the ducts, as well as that lining almost the entire gastric gland, does not show any pronounced alteration except at the terminal portion of the fundus, where the cytoplasm of the cells is decidedly granular. The mucosa and submucosa are hypertrophied and somewhat congested. The most characteristic feature of this section is the excessive and diffused proliferation of the connective-tissue cells, accumulated in greatest quantity at the bottom of the mucosa and extending along the course of the gastric glands to the surface of nearly every tubule. The proliferating masses of this inflammatory cell infiltration are intermingled with a considerable amount of polynucleated, polymorphonucleated leucocytes and eosinophiles which are readily distinguished by their staining fromthe connective-tissue cell proliferation. The muscularis, both circular and longitudinal portions, is slightly hypertrophied, but shows no other alteration. The serous coat appears unaffected.

Slide B.—Section of kidney comprising cortex and medulla. Nearly all the capillaries of the cortex are overdistended by blood; there are also extensive outwandering of leucocytes and diapedesis of the red blood corpuscles between the convoluted tubules of the labyrinth and around and within the capsule of Bowman. There are also several small hemorrhagic areas in different portions of the cortex. The cytoplasm of the renal epithelium in the proximal and distal convoluted tubules is quite granular and in different stages of disintegration. Some of the tubules show very pronounced degeneration and some desquamation. The nuclei of these cells are also in various stages of degeneration, and while some have taken the staining quite well, others are entirely blended with the cytoplasm of the cell. The medulla shows excessive congestion, but no desquamation of the epithelium.

Slide C.—Section of lung. The pleura is thickened. The pulmonary capillaries are distended, but there is no pronounced outwandering of leucocytes and no diapedesis of the red blood corpuscles. A small amount of catarrhal exudate is present in some of the alveoli, but not sufficient to cause any pronounced disturbance. There is some cell proliferation around the bronchial tubes and an increase in the interlobular connective tissue.

Slide E.—Section of heart. The individual cardiac fibers are not perceptibly altered by degeneration, though the capillaries are noticeably distended, a considerably amount of outwandered leucocytes and diapedesis of red blood corpuscles and several circumscribed small hemorrhagic areas are present.

Slide F.—Section of small intestines. The epithelium covering the villi appears unimpaired, that lining Lieberkuhn's glands swollen, decidedly granular but not desquamated. The mucosa as well as the submucosa is considerably hypertrophied, and all the capillaries are distended, indicating a pronounced congestion. The most characteristic feature of this preparation is the excessive diffuse inflammatory cell proliferation, differing very materially from the more compact and well circumscribed cell aggregations of the solitary glands and Peyer's patches. Both the circular and longitudinal muscular coats are hypertrophied and more loosely arranged than normally. The intermuscular plexus of Auerbach is greatly enlarged, and some of the ganglion cells are very granular. One part of the section shows also a well-defined Peyer's patch.

Case 12

Slide A.—Section of stomach. There is no erosion or necrosis on the surface of the mucosa, which is greatly hypertrophied and intensely congested, as indicated by the overdistended capillary vessels and severe hemorrhagic areas present in the mucosa, submucosa, and muscularis. The epithelium lining the ducts of the gastric glands is not altered, while that of the parietal and the chief cells is quite granular, degenerated, and desquamated in most of the peptic glands.

Slide B.—Section of kidney comprising cortex and medulla. The capsule is slightly thickened and adherent. All the capillaries are congested, especially those of the glomeruli. There is a considerable number of leucocytes, decided diapedesis between the convoluted tubules of the labyrinth, also several large hemorrhagic areas in several portions of the cortex. The medulla is even more congested than the cortex. The renal epithelium lining the proximal and distal

convoluted tubules is granular and degenerated, but shows no marked necrosis, nor is it desquamated.

Slide C.—Section of lung. There is an excessive congestion of the pulmonary capillaries, accompanied by outwandered leucocytes and diapedesis of red blood corpuscles and numerous small hemorrhagic areas. The interalveolar, peribronchial, and interstitial connective tissues show a moderate amount of cell proliferation. There is also a small amount of catarrhal exudate in the infundibuli, but no extensive desquamation of the pulmonary epithelium.

In addition to these slides described above, many slides prepared by Dr. D. E. Salmon were examined by the writer, as well as a number of slides prepared under the direction of Dr. V. A. Moore from animals experimentally poisoned.

CONCLUSIONS FROM MICROSCOPIC EXAMINATIONS

The microscopic changes in these tissues are as characteristic as the post-mortem appearances in any smelter district, and a careful study of the preparations, supported in most instances by a chemical analysis, can be summed up as follows: (1) Vascular changes, (2) epithelial changes, (3) connective-tissue cell proliferation.

1. Vascular changes, which varied from slight dilatation, fullness, and distention of the capillaries in the stomach, intestines, kidneys, liver, lungs, and heart to more pronounced conditions accompanied by outwandering of leucocytes, diapedesis of red blood corpuscles, and in some instances hemorrhagic areas varying in size and corresponding to the petechiæ seen on macroscopic inspection. The pronounced congestion of the gastrointestinal tract, but particularly of the stomach, is evidently due to the action of an irritant that has brought about similar conditions in other organs concerned in elimination and metabolism, namely, the kidneys, liver, and lungs. The degree of these changes varies in intensity—acute, in Doctor Moore's experimental cases of arsenic poisoning, subacute and chronic in Doctor Salmon's cases and in those described above. Chemical analysis proved the irritant to be arsenic.

2. Epithelial changes vary from a slight granular condition of the cytoplasm to a marked degeneration of the cytoplasm and from a breaking down and vacuolation to a destruction and loss of cell substance, which in the advanced stages of metabolism is the forerunner of fatty degeneration. It is also accompanied by changes in the karyoplasm, as fragmentation or dissolution of the chromatin. This is demonstrable by staining; when the chromatin is but slightly affected the nuclei take the staining quite well, but stain fainter in proportion to the dissolution of the chromatin and become finally invisible by the completion of the process of karyolysis, when the karyoplasm apparently has blended with the cytoplasm. These changes are most pronounced in the organs of metabolic activity—the liver and kidneys—but may also be observed to a less degree in the gastric cells. There is also desquamation of the epithelium in the proximal and distal convoluted tubules, and to a less degree in the collecting tubules of the kidney and the respiratory epithelium of the air cells.

3. The connective-tissue cell proliferation or multiplication, which can only be the result of an irritant, inflammatory in character, is well pronounced in the mucous membrane in the stomach, the name of gastroadenitis parenchymatosa arsenicalis having been suggested by Virchow for similar cell proliferations in the stomach. The cell proliferation is present at the bottom of the mucosa and frequently extends toward the surface, compressing the fundus and the gastric

glands. It is most marked in the vicinity of the blood vessels, but extends as a loose accumulation throughout the mucosa, differing very materially from the denser and more circumscribed so-called lenticular glands which are the lymph nodes normally present in the mucosa of the stomach and the solitary glands of the intestines. This cell proliferation was not limited to the gastrointestinal tract, but was present also in the cortex of the kidney between the convoluted tubules, in the liver in the periportal areas, and in the lung around the bronchi in the peribronchial tissue.

GENERAL SUMMARY

1. The Washoe smelter, near Anaconda, Mont., is situated on a high hill towering above the Deer Lodge Valley, which extends 30 miles in length and 4 to 6 miles in breadth. The smelter roasts daily from 8,000 to 10,000 tons of ore which is quite rich in arsenic. The dense volume of smoke containing smelter fumes and flue dust rises high under favorable atmospheric conditions and can be traced for over 16 miles as a continuous cloud to its origin, the huge smokestack, 300 feet high and 30 feet wide at the orifice. Prevailing aerial currents direct the course of the smoke to various parts of the valley, the foothills, and mountains. The great heat at which the fumes and flue dust are generated, aided by the forced draft, is responsible for the excessive velocity of the contaminating particles, which, after cooling in the air, are precipitated and deposited not only on the grasses of the valley, but also on the vegetation of the foothills and the foliage of the surrounding forests. The emanations of flue dust from the smelter contain from 22 to 30 tons of arsenic every twenty-four hours.

2. Damage to grasses and forests can be detected even by an untrained casual observer, and thorough investigation by expert foresters and chemists has proved the existence of extensive damage. The soil, when irrigated by water that is contaminated by the waste products of the smelter which reach some of the streams, suffers deterioration of its fertility, as is shown by the diminution of the crops.

3. The chemical analyses of the grasses, hay, and soil by Dr. J. K. Haywood and by Professors Harkins and Swain show the presence of a considerable amount of arsenic. According to Harkins and Swain, arsenic is found not only in the above-mentioned substances, but also in the hair, stomach, intestines, liver, pancreas, spleen, brain, spinal cord, thyroid gland, muscles, lungs, bones, heart, bladder, salivary glands, fat, adrenals, and blood of the animals in that vicinity. This includes not only all the vital organs, but practically every structure in the animal economy.

4. Animals pasturing in Deer Lodge Valley become unthrifty and suffer from digestive disturbances and derangements of the organs concerned in the assimilation of food, and death frequently occurs in the endeavor to eliminate the irritating substances from their systems. Those that survive are rendered worthless for breeding purposes. Cattle intended for beef are too lean to be marketable, while horses are rendered unfit for work or driving purposes.

5. The pathologic findings on post-mortem examination show unmistakable lesions of chronic catarrhal inflammation of the stomach, intestines, lungs, and kidneys. The microscopic preparations demonstrate conclusively the presence of a pronounced irritant, which has

operated on all organs pertaining to the metabolism and assimilation of food, as well as those concerned in the elimination of waste products, being characterized by an inflammatory cell proliferation in the mucous membrane of the stomach and intestines, the portal areas of the liver, the peribronchial connective tissue of the lung, and the interstitial connective tissue of the kidneys. These cell infiltrations or proliferations can be the result only of an irritant, which on chemical analysis has proved to be arsenic.

6. From the foregoing we conclude that smelter fumes and flue dust, laden as they are with highly dynamized mineral elements, form a most certain means of distributing such elements over regions of greater or less extent; that where smelter smoke and flue dust contain arsenic the amount of that toxic mineral distributed in the course of a day reaches many tons; that careful investigations of the region affected by smelter smoke reveal a steadily growing diminution in the quantity and quality of crops and vegetation of all kinds; that the same kind of deterioration is noticeable in the livestock industry, large numbers of horses and cattle and other stock dying, and those that continue to live manifesting lowered vitality, depraved nutrition, and derangements of the breeding function even to sterility; that both on the Continent of Europe, and particularly in the Deer Lodge Valley of this country, where the writer's investigations were conducted, the clinical symptoms of animals thus affected coincide with those known to science as symptoms of arsenical poisoning; that the post-mortem findings in animals that have died or in affected animals purposely killed confirm such diagnosis; that the histologic examinations of the tissues of such animals abundantly establish the accuracy of this diagnosis; that feeding experiments with known quantities of arsenic and the experimental local application of arsenic resulted in the production of symptoms and lesions identical with those found in affected animals; that expert chemical investigations, not only of the grasses, hay, soil, etc., but also of all animal tissues, demonstrate the presence of toxic quantities of arsenic; that finally we have in the Deer Lodge Valley an industry which under present conditions is wholly incompatible with agriculture, destructive alike of animal and vegetable life.

BIBLIOGRAPHY

Bevan, L. E. W. Poisoning of stock with arsenic. Veterinary Journal, vol. 64, No. 401, pp. 557–559. London, Nov., 1908.

Borggreve, B. Waldschäden im oberschlesischen industriebezirk nach ihrer entstehung durch Hüttenrauch, Insektenfrass. Eine Rechtfertigung der industrie gegen folgenschwere falsche anschuldigungen. Frankfurt, 1895.

Ueber das Robert Hartig'sche Kennzeichen der Rauchbeschädigung an fichten. Zeitschrift für Forst- und Jagdwesen, Jahrg. 29, pp. 158–176. Berlin, 1897.

Braconnot, H., and Simonin, F. Notes sur les émanations des fabriques de produits chimiques. Journal de Chimie Médicale, de Pharmacie et de Toxicologie, 3d ser., tome 4, pp. 280–288. Paris, 1848.

Also in Dingler's Polytechnisches Journal, Band 108, pp. 264–270. Stuttgart, 1848.

Cameron, S. S. Diseases of farm animals. Mineral poisons. The Journal of the Department of Agriculture of Victoria, vol. 7, pt. 1, pp. 47–53. Melbourne, Jan., 1909.

Chatin, Ad. Etudes de physiologie végétale faites au moyen de l'acide arsénieux. Comptes Rendus de l'Académie des Sciences, tome 20, pp. 21–29. Paris, 1845.

Danger and Flandrin. On the effects of arsenic on sheep. Veterinarian, vol. 16, pp. 345–346. London, 1843.

DANGLER, L. Über die Einwirkung des Rauches auf die Vegetation. Hannoversche Land- und Forstwirtschaftliche-Zeitung. No. 22. Wien, 1887.

DAVY, EDMUND W. On the presence of arsenic in some artificial manures, and its absorption by plants grown with such manures. The London, Edinburgh and Dublin Philosophical Magazine and Journal of Science, 4th ser., vol. 18, No. 118, pp. 108–113. London, Aug., 1859.

FREYTAG, MORITZ. Wissenschaftliches Gutachten über den Einfluss welchen die Hüttenwerke der Mansfelder Kupferschiefer bauenden gewerkschaft in dem Wipperthal zwischen Mansfeld und Hettstedt auf die Vegetation der benachbarten Grundstücke und indirekt auf Menschen und Tiere ausüben. Eisleben, 1870.

Wissenschaftliches Gutachten über den Einfluss welchen die Hüttenwerke der Mansfelder Kupferschiefer bauenden gewerkschaft in dem Wipperthal zwischen Mansfeld und Hettstedt während des Jahres 1870 auf die Vegetation der benachbarten Grundstücke und indirekt auf Menschen und Tiere ausüben. Eisleben, 1871.

Wissenschaftliches Gutachten über den einfluss des Hüttenrauchs bei den fiscalischen hüttenwerken zu Freiberg auf die Vegetation der benachbarten Grundstücke und ganz besonders auf die Gesundheit der Haustiere, namentlich des Rindvieh's. Jahrbuch für das Berg- und Hüttenwesen im Königreiche Sachsen auf das Jahr 1873. Abhandlungen aus dem gebiete des Berg- und Hüttenwesens, pp. 3–73. Freiberg.

Zweites Gutachten über den Einfluss des Hüttenrauchs bei den fiskalischen Hüttenwerken zu Freiberg auf die Vegetation der benachbarten Grundstücke und ganz besonders auf die Gesundheit der Hausthiere, namentlich des Rindvieh's. Jahrbuch für das Berg- und Hüttenwesen im Königreiche Sachsen auf das Jahr 1875. Abhandlungen aus dem gebiete des Berg- und Hüttenwesens, pp. 3–54. Freiberg.

Die schädlichen Bestandtheile des Hüttenrauchs der Kupfer-, Blei-, und Zinkhütten und ihre Beseitigung. Landwirtschaftliche Jahrbücher, Band 11, pp. 315–357. Berlin, 1882.

FRICKE, E. Beschädigung von Garten- und Feldgewächsen durch Hüttenrauch. Die Landwirtschaftlichen Versuchs-Stationen, Band 34, pp. 277–283. Berlin, 1887.

GORUP-BESANEZ, E. VON. Ueber das Verhalten der Vegetirenden Pflanzen und der Ackererde gegen Metallgifte. Annalen der Chemie und Pharmacie, Band 127, Heft 2, pp. 243–256. Leipzig und Heidelberg, 1863.

GREAT BRITAIN. First Report of the Royal Commission on Arsenical Poisoning, part 1. London, 1901.

Local Government Board, Alkali, etc., Works. Annual reports of the chief inspector for the years 1902–1906. London.

Report and Minutes of Evidence taken before the Royal Commission on Noxious Vapours, London, 1878. (Abstract in Chemical News, vol. 38, pp. 181–182. London, Oct. 11, 1878.)

HARKINS, W. D., and SWAIN, R. E. The determination of arsenic and other solid constituents of smelter smoke, with a study of the effects of high stacks and large condensing flues. 1st paper. Journal of the American Chemical Society, vol. 29, No. 7, pp. 970–998. Easton, Pa. July, 1907.

Arsenic in vegetation exposed to smelter smoke. 2d paper. Journal of the American Chemical Society, vol. 30, No. 6, pp. 915–928. Easton, Pa. June, 1908.

The chronic arsenical poisoning of herbivorous animals. 3d paper. Journal of the American Chemical Society, vol. 30, No. 6, pp. 928–946. Easton, Pa. June, 1908.

HARTIG, ROBERT. Ueber die Einwirkung des Hütten- und Steinkohlenrauches auf die Gesundheit der Maldelwaldbäume. Forstlich-Naturwissenschaftliche Zeitschrift, Jahrg. 5, Heft 7, pp. 245–290. München, 1896.

HASSELHOFF E., and LINDAU, G. Die Beschädigung der Vegetation durch Rauch. 412 pp. Leipzig, 1903.

HAUBNER. Die durch Hüttenrauch veranlassten Krankheiten des Rindviehes im Hüttenrauchsbezirke der Freiberger Hütten. Archiv für wissenschaftliche und practische Thierheilkunde, Band 4, Heft 2/3, pp. 97–136; Heft 4/5, pp. 241–260. Berlin, 1878.

HAYWOOD, J. K. Injury to vegetation by smelter fumes. U. S. Department of Agriculture, Bureau of Chemistry, Bulletin 89, Washington, 1905.

Injury to vegetation and animal life by smelter wastes. U. S. Department of Agriculture, Bureau of Chemistry, Bulletin 113, Washington, 1908.

HERING, C. A. Die Verdichtung des Hüttenrauches. 2d auf. Stuttgart, 1888.

HUSSON, C. Action de la fumée des fours à chaux sur les vignes. Comptes Rendus de l'Académie des Sciences, tome 82, pp. 1218–1221. Paris, 1876.

JÄGER, GEORG F. VON. Ueber die Wirkungen des Arseniks auf Pflanzen. 116 pp. Stuttgart, 1864.

KÖNIG, J., STEFFECH, and HEINE, H. Der Schutz gegen Flurschädigungen durch gewerbliche Einwirkungen. Arbeiten der Deutschen Landwirtschafts-gesellschaft, Heft 14, pp. 1–44. Berlin, 1896.

LÜTY, FR. Beschädigung der Vegetation durch Hüttenrauch. Zeitschrift für angewandte Chemie, Heft 5, pp. 163–164. Berlin, March 1, 1891.

MARCET, F. De l'action des poisons sur le règne végétal. Annales de Chimie et de Physique, tome 29, pp. 200–223. Paris, 1825.

MERBACH, KURT. Die Anlagen zur Unschädlichmachung des Rauches auf den fiscalischen Hüttenwerken bei Freiberg. Jahrbuch für das Berg- und Hüttenwesen im Königreiche Sachsen auf das Jahr 1881, pp. 42–49. Freiberg.

OST, H., WEHMER, C. Zur Beurtheilung von Rauchschäden. Die chemische Industrie, Jahrgang 22, No. 11, pp. 233–237. Berlin, June 1, 1899.

PERCY, JOHN. Metallurgy. 634 pp. London, 1861.

PFÜTZNER. Schädliche Einwirkung des Steinkohlen- und Hüttenrauches auf die holzvegetation. Jahrbuch des Schlesischen Forst-Vereins für 1881, pp. 33–38. Breslau, 1882.

PREVOST, E. W. Beiträge zur Kenntnis der Beschädigung der Pflanzen und Bäume durch Hüttenrauch. Die landwirtschaftlichen Versuchs-Stations, Band 35, pp. 25–28. Berlin, 1888.

RAMANN, E. Ueber Rauchbeschädigungen. Zeitschrift für Forst- und Jagdwesen, Jahrgang 28, Heft 9, pp. 551–556. Berlin, Sept., 1896.

REICH, F. Ueber die bei aussersächsischen Hüttenwerken beobachteten Wirkungen des Hüttenrauches und die dagegen ergriffenen Massregeln. Jahrbuch für den Berg- und Hüttenmann, pp. 127–163. Freiberg, 1867.

REUSS, CARL. Hüttenrauchschaden in den Waldungen des Oberharzes. Dingler's Polytechnisches Journal, Jahrgang 241, pp. 124–132; 204–212; 285–291. Augsburg, 1881. Also in Zeitschrift für Forst- und Jagdwesen, Jahrgang 13, Heft 2, pp. 65–91. Berlin, Feb., 1881.

Gutachten über Rauchbeschädigung in dem von Tiele-Wincklerschen Forstreviere Miechowitz-Rokittnitz. Goslar, 1893.

Rauchbeschädigung in dem von Tiele-Wincklerschen Forstreviere Myslowitz-Kottowitz, 236 pp. Goslar, 1893.

ROSLER. Ein Beitrag zu den Beobachtungen über die schädlichen Einflüsse des Hüttenrauches auf Pflanzen und Thiere. Mittheilungen des landwirtschaftlichen Institutes der Universität. Halle, 1865.

SCHROEDER, JULIUS VON. Ueber die Beschädigung der Vegetation durch Rauch, eine Beleuchtung der Borggreve'schen Theorien und Anschauungen über Rauchschäden. 35 pp. Freiberg, 1895.

SCHROEDER, JULIUS VON, and REUSS, CARL. Die Beschädigung der Vegetation durch Rauch und die oberharzer Hüttenrauchschäden. 333 pp. Berlin, 1883.

SCHROEDER, J. VON, and SCHERTEL, A. Die Rauchschäden in den Wäldern der Umgebung der fiscalischen Hüttenwerke bei Freiberg. Jahrbuch für das Berg- und Hüttenwesen im Königreiche Sachsen auf das Jahr 1884, Heft 2, pp. 93–120. Freiberg, 1884.

SCHROEDER, VON, and SCHMITZ-DUMONT, W. Neue Beiträge zur Rauchfrage. Tharander Forstliches Jahrbuch, Band 46, pp. 1–57. Dresden, 1896.

STOECKHARDT, A. Untersuchungen über die schädliche Einwirkung des Hütten- und Steinkohlenrauches auf das Wachsthum der Pflanzen, insbesondere der Fichte und Tanne. Tharander Forstliches Jahrbuch, Band 21, pp. 218–254. Dresden, 1871.

SWAIN, R. E., and HARKINS, W. D. Arsenic in vegetation exposed to smelter smoke. 2d paper. Journal of the American Chemical Society, vol. 30, No. 6, pp. 915–928. Easton, Pa. June, 1908. (See also Harkins and Swain.)

TURNER, EDWARD, and CHRISTISON, ROBERT. On the effects of poisonous gases on vegetables. Edinburgh Journal of Science, o. s. vol. 8, No. 1, pp. 140–149. Edinburgh, Jan., 1828.

VATER. Der Nachweis von Rauchschäden durch die chemische Analyse. Tharander Forstliches Jahrbuch, Band 47, pp. 254–263. Dresden, 1897.

WISLICENUS, H. Zur Beurtheilung und Abwehr von Rauchschäden. Zeitschrift für angewandte Chemie, Jahrg. 14, Heft 28, pp. 689–712. Berlin, 1901.

O